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EDITORIAL

WILLIAM HAZLITT once remarked that when a thing ceases to be a subject of controversy, it ceases to be a subject of interest. When the fervour of debate has passed, order, and not of necessity a new order, settles into place. The contentions of yesterday become little more than the diversions of today, and propositions which rent the calm of societies pass into obscurity, and then oblivion. But from it all ideas acquire cohesion, facts assemble, and knowledge takes a step forward. Such a course, variously punctuated, forms the story of all achievement; and the history of medicine is replete with illustrations.

Primary tuberculosis is a subject now debated in this way, for at present it bristles with controversial matter. The remarkable innocence of most primary infections is accepted, and their frequency in childhood too. Yet beyond this age a primary infection has been thought to take a more dangerous course, for in contrast to the latency in youth it then leads more often to active and progressive disease. Until recently the same was held for infancy, but the detailed studies of Miriam Brailey have shown that white children, under two years of age, at any rate show a high rate of survival after such infection. Before conclusions can be drawn that the reaction to a primary infection in adult life differs from that of childhood we need to know more of the eventual outcome in adults, and also of the clinical course which their primary lesion induces. Until now there has been little said on this side of the problem, and so far it is not established that such disease differs greatly from that of childhood, even though it may be more a clinical than radiological and allergic entity.

Another and equally important, if depressing, aspect of primary infection is the bearing which it has upon resistance to subsequent fresh exposure to the disease. As Hall puts it in his stimulating paper in this issue, is the

primarily infected human being "immune" or is he "sensitive"? Prior infection conveys some immunity to reinfection, though admittedly it may be limited. But the paradox then arises that once reinfection occurs the outcome is seen as disabling disease in a sense unknown with the majority of primary lesions. There is an inherent assumption here, that it minimises the significance of the host of primarily infected persons who meet reinfection and yet never show that they have done so by any recognised test; and there are many others who do show such evidence, when perchance they come our way, and yet have remained healthy. None the less there is much to favour the view that primary infection fails to protect against reinfection. Stewart goes further, for he regards the primary as the initiator of changes without which destructive forms of tuberculosis hardly occur, a view which Hall supports.

These changes, comprised in the term allergy, certainly alter the aspect of the disease, and there are many ways in which they can rightly be regarded as detrimental to the interest of the patient. But we know that allergy appears quickly after tuberculous infection, as a rule within a few months, so that the individual infected for the first time in adult life is bound to acquire the disadvantages of such reaction within a measurable time of infection. The view that a previous primary infection has an exclusive, and often distant, influence thus implies that the course of events within quite a brief period goes far to decide the issue of a benign or malignant outcome, a sinister inference so far as treatment is concerned. Whatever the truth there is here much food for thought, and Hall has reviewed the present state of knowledge in a clear and impartial way that throws into relief the immense difficulties of the problems.

C. H.

GENERAL ARTICLES

PREVENTION OF TUBERCULOSIS IN WAR-TIME

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The fundamental principles of the prevention of tuberculosis remain the same in war as in peace. The etiology of the disease and the characteristics of the tubercle bacillus are unchanged by the politics of nations. War does, however, change environment and living conditions, thereby disturbing the balance of forces involved in the struggle to overcome the spread of the infection. The subject under discussion, therefore, resolves itself into a study of war-time conditions with a view to instituting measures which will counteract the influence of those conditions which favour the development of tuberculosis.

In the present war, four main features have a special bearing on public health. These are evacuation, air-raid precautions, rationing of food and redistribution of labour. To what extent do these favour or prevent tuberculosis?

Evacuation.—One would have thought that the movement of large masses of the population would have scattered carriers of tuberculous infection amongst healthy folk and thereby tend to increase the incidence of the disease in the reception areas. It is too early to draw definite conclusions, but so far there is no evidence to support this supposition. This is due to the fact that the large majority of evacuees under the Government scheme were healthy and those who might have been in any way debilitated or in a pre-tuberculous condition have benefited from the change to rural surroundings and have improved considerably in general health. There are, however, a number of individuals suffering from active phthisis who have moved into the country on their own accord, in some cases to unsatisfactory billets. As soon as the presence of these dangers is known it is essential that every effort is made to segregate the tuberculous individuals and to persuade the contacts to be medically examined. Unfortunately in many reception areas there is a shortage of beds for tuberculous evacuees. The difficulty can be partially overcome by a reciprocal arrangement between the local

authorities of the reception and evacuation areas whereby provision for treatment of evacuees is made by the local authority of the evacuation area and *vice versa*. Owing to the reduction in the population in the evacuation area, it is probable that sanatoria serving such areas will have vacant accommodation. The danger of healthy children being billeted in tuberculous households has had the closest attention of tuberculosis officers in all reception areas and should not be possible under any well-organised official scheme, although constant vigilance is needed to prevent catastrophes.

Air-Raid Precautions.—Although evacuation tends to separate the population, aerial bombardment tends to bring individuals into closer contact with each other. The " black out " keeps families indoors under poor conditions of ventilation, crowded air-raid shelters produce a conglomeration of people in an environment favourable to cross-infection, loss of sleep and inadequate rest cause fatigue and predispose to general lowering of resistance to disease. This is often increased by A.R.P. duties superimposed upon a heavy day's work, probably with overtime. Bombed-out families are thrown together in rest homes and hostels where contact with active tuberculous cases is difficult to locate and manage. Daily routine is disorganised, whilst work is harder and meals often inadequate and taken hastily. Added to all these is the anxiety and depression which war conditions exert both consciously and unconsciously on the majority of middle-aged and old people. All these factors favour the development of tuberculosis, but on the other hand many are earning more money and there is less indulgence in gaiety and strenuous pleasures. Some also have actually benefited by a complete change in work and living conditions, and with a regularly planned life have improved both physically and mentally.

Rationing of Foodstuffs.—During recent pre-war years there has been no shortage of the protective foods. Even though certain sections of the people may have had difficulty, through financial reasons, in obtaining adequate quantities of first-class proteins, there has been always a plentiful supply of fruits, vegetables, eggs, cheese, milk and fish. These are now becoming scarcer and more expensive. It is probable that the good supply of these commodities in the past, particularly in rural areas, has contributed in no small measure to the decline in the incidence of tuberculosis. It certainly has been responsible for the good health of individuals in otherwise unhygienic surroundings. It may be expected, therefore, that the gradual elimination of these foodstuffs will reduce the general resistance to infection, and attention should be paid to relieving the deficiency, particularly in sections of the community most susceptible to tuberculosis.

Not only is man being rationed, but cattle also are receiving a limited supply of their usual fodder, particularly during winter months. This may

have a serious effect on their health, and as about 40 per cent. of stock in this country were known to be infected with tuberculosis it would not be surprising to find an increase in the number of milk-producing cows suffering from active disease in the future. Compulsory and efficient pasteurisation of all milk is necessary to deal with this menace.

Redistribution of Labour.—This is meant to be taken in its widest sense and to include the change of occupation due to recruitment to the services. Many have been conscripted to the Army, Navy and Air Force; others have found new occupations in munition work or on the land. These changes in work and environment will affect the individual's chance of contracting tuberculosis. Broadly speaking it may be assumed that, unless there is active disease present or direct contact with massive infection, the individual will be protected against the development of tuberculosis where the change in living conditions insures food sufficient to cover the energy output, regular routine and adequate rest, whereas his liability to tuberculosis will be increased if there is fatigue, irregular hours, anxiety and lack of the essential foods. Joining the services will therefore, in healthy individuals, tend to reduce the incidence of tuberculosis by giving the necessary improvement in physical fitness just to those ages most susceptible to the disease.

Many doctors and nurses have given up their usual routine and are entirely engaged on war work, whilst those remaining have had to devote a considerable period of their time to A.R.P. duties, so reducing the attention they can give to the diagnosis and treatment of the usual peace-time epidemics and disabilities. This disorganisation of the medical services of the country has an influence on the spread of tuberculosis. If there are insufficient medical and nursing staffs to operate dispensaries and to commission beds for treatment and segregation of active tuberculous patients, a number of these cases will be free to act as sources of infection amongst a healthy population living under considerable mental and physical strain.

The change of occupation to munition work will benefit the household and the individual by bringing more money to the family, but this advantage to actual workers may be counterbalanced by fatigue and unwise spending.

From this brief summary of the peculiar conditions brought about by war it will be seen that some factors favour the spread of tuberculosis whilst others definitely help to protect the community against the disease.

War and the Incidence of Tuberculosis.—It is unwise to generalise on the effect of the war on the incidence of tuberculosis without first assessing the relative influence exerted by each new war condition. This is difficult whilst the war is proceeding, as one cannot see wood for trees, but some help can be obtained from a study of the effect of the last Great War on tuberculosis mortality figures.

The following table gives the mortality figures for tuberculosis (all forms) per 100,000 population in countries from 1911 to 1930:

Year.	England and Wales.	Scotland.	France.	Belgium.	Germany.	U.S.A.	Italy.	Holland.	Denmark.	Spain.	Sweden.	Norway.
1911	147	171	127	127	158	159	149	158	133	157	191	221
1912	137	172	211	118	156	150	149	154	136	148	195	221
1913	135	169	213	118	142	148	145	142	126	152	185	220
*1914	136	162	233	124	139	147	158	140	131	155	193	225
*1915	151	163	227	131	148	146	167	144	125	160	204	219
*1916	153	159	219	149	155	142	166	167	138	164	206	226
*1917	162	157	266	211	203	146	175	189	158	171	195	207
*1918	169	158	278	245	228	150	209	203	128	201	176	194
1919	126	129	209	144	208	126	173	174	114	178	164	190
1920	113	124	185	114	152	114	160	147	113	186	162	210
1921	113	117	193	110	136	99·4	142	127	97·6	157	156	198
1922	112	119	—	111	142	96·8	139	114	95·8	155	151	201
1923	106	118	—	106	149	93·6	143	106	89·9	155	138	190
1924	106	116	—	103	119	90·4	152	106	98·1	154	140	200
1925	104	110	160	100	107	88·6	150	99·1	92·4	152	150	188
1926	96·4	99·1	166	95	97·7	87·1	144	97	81·3	151	132	169
1927	97·1	99·5	175	97	93	80·8	134	94·3	78·5	147	136	170
1928	93	96·6	167	—	88	79	—	83·8	75	138	—	156
1929	96	94	168	—	—	—	—	—	—	135	—	—
1930	—	—	—	—	—	—	—	—	—	—	—	—
	Belligerents							Non-belligerents				

Figures from the Service de la Statistique du Comité National.
* War period.

The outstanding features of these figures are the direct relationship of the rise in mortality to the degree of involvement in the war and also the dramatic decrease in mortality in all countries except Norway after 1918. Countries like the United States and Scotland, although belligerents, did not feel the effects of the war, neither did the distant countries of Norway and Sweden. In all these places there was either no change or an actual decrease in the mortality from tuberculosis. Holland and Spain, on the contrary, were considerably affected by the war and show the same rise as in belligerent countries. Professor Collis[1] has shown that the sudden fall in the mortality curves after 1918 was mainly due to the return to an adequate diet. Two other factors would also contribute in a lesser degree to the subsequent lowering of the death rate from tuberculosis. The world-wide influenza epidemic of 1918 removed many infectious cases and the battle fronts reduced the number of young male adults in those age groups

most susceptible to tuberculosis. The rise and fall of the mortality curve during the war period was most marked between the ages of fifteen and thirty-five, which were also the ages most severely hit by the influenza epidemic, and in the cases of females the ages most frequently absorbed into industry. The influence of industrialism, particularly in females, has been stressed by D'Arcy Hart.[2]

There is no doubt that diet and industrialisation are closely connected with the incidence of tuberculosis, and it is known that the nutrition of all workers should be adequate for and bear a direct relation to their energy output if good health is to be maintained. At the present time the happy equilibrium of 1940 is likely to be disturbed by the increase of stress and decrease of food, thereby creating a weak link in the defensive chain controlling and checking epidemics and also tuberculosis.

Preventative Measures already Taken.—The Ministry of Health have had tuberculosis under close consideration since the outbreak of hostilities. The Horder Committee and the Standing Advisory Committee have made recommendations on the majority of the major problems connected with shelter accommodation, recruitment, mass radiography and evacuation. It has not been possible to put all these suggestions into practice owing to the difficulty of introducing new technique and organisation during intense war activities.

An effort has been made to remove the infectious cases from public shelters, and compulsory powers have been given to assist local authorities in the work. More attention has been given to eliminating the tuberculous from the Services, although mass radiography has only been practised to a very limited extent and has not been recommended as a general measure. Financial assistance has been assured to local authorities who find themselves burdened with tuberculous evacuees, but extra personnel to deal with the increase in dispensary and sanatorium work is difficult to obtain; neither has the problem of extra bed accommodation for segregation and treatment purposes been solved.

Contact examinations have decreased and a good number of unemployed tuberculous positive cases have found work with good remuneration, but not always suitable work under satisfactory conditions. Except for additional milk, special consideration of the tuberculous has not been thought necessary in food-rationing schemes, neither has there been any suggestion of granting extra nourishment to direct contacts of infectious cases or to tuberculosis workers. The control of catarrhal diseases is being given much attention, and should help to reduce the spread of tuberculosis by droplet injection, but stronger measures are required to stop the growing habit of promiscuous spitting.

Discussion.—It will be seen that there are serious gaps in our defences against tuberculosis. From the experience gained during and after the first Great War, it may be assumed that the three main weapons are adequate nutrition, sufficient rest and control of carriers of the infection. None of these is easy to ensure during hostilities, and it must be emphasised that war-time is not the period to experiment widely with new methods or to establish new organisations for the prevention of tuberculosis. Although plans may be drawn up for post-war reorganisation during the struggle, it is better to develop existing services where needed to meet the particular circumstances and to take general measures to protect the health of the community on the fundamental principles of physiology and hygiene. The labourer is worthy of his food and rest. Extra nourishment, particularly in the protective foods, should be granted in proportion to the energy output of the worker. This could be done on a graduated scale of rations, as has been done with the cheese issue to agricultural labourers. Those in direct contact with individuals suffering from tuberculosis should also have special consideration. A certain amount of propaganda on how to spend leisure time and means of securing sufficient rest would be useful, particularly to young adults.

The policy of segregation demands that there must be accommodation to receive the segregated. At present they tend to block beds needed for the reception of those with early or treatable lesions. Provision of hostels and development of the colony system are methods by which the problem can be solved. It is important that individuals showing signs or symptoms of active tuberculosis should be excluded from the Services. An efficient though costly way of meeting the difficulty would be to submit all recruits to mass radiography, but the impracticability of initiating such facilities in war-time have been realised. Much, however, could be done by more thorough sputum examinations, greater attention to clinical histories and more consultations with tuberculosis specialists. The search for the hidden case becomes increasingly important in war-time, and examination of contacts should be encouraged. This means that the rôle of the dispensary should be enlarged rather than decreased, and its services linked up more closely with industrialism and the fighting forces. It must not be forgotten that the diagnosis of tuberculosis often requires specialised knowledge, and the training of students in this work should not be neglected in war-time.

Milk has always been closely connected with tuberculosis problems, both as a means of spread and prevention. The shortage which is likely to occur in the future will be difficult to overcome by substitutes, and it is probable that adults will have to forgo their usual quantity to provide for infants and young children. To prevent tuberculous infection, all milk should be pasteurised before sale. The distribution of milk along with

other essential foods might be subject to graduated rationing—*e.g.*, points calculated to meet the special needs of particular groups of the population.

Conclusion.—The spread of tuberculosis may become during the war one of the hidden dangers of national importance. Much can be done to prevent this occurring by raising the standard of employment conditions and distribution of essential foods to those running a special risk of contracting the infection. Proper use of leisure time and regular hours of work and sleep will do much to overcome fatigue. Finally, and probably most important of all, is the control of sources of infection by segregation, pasteurisation of milk, education and the suppression of catarrhal infections. Our knowledge of the etiology of tuberculosis is not complete, but we know sufficient about the direct and predisposing causes to take action. Let us do so before it is too late.

The views expressed in this article are the personal opinions of the author, and do not necessarily represent those of the London County Council.

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SOME THOUGHTS ON THE PROBLEMS AND PARADOXES OF PRIMARY TUBERCULOSIS

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ALMOST every practising physician must have found it difficult, at some time or another, to give anxious parents a satisfactory explanation of the presence of a positive tuberculin reaction and a well-defined primary tuberculous complex in one child and the absence of one, or both, in another. Both children are healthy, but which is the better equipped to face the dangers of life for which it is being prepared? A correct understanding and explanation of the nature and effects of primary infection of the human being with the tubercle bacillus seems to demand the solution of many complicated paradoxes.

NATURAL RESISTANCE.—The fact that the ordinary course of life in civilised countries results in the infection of a majority of persons before they reach adult life suggests that man is an animal who is extremely susceptible

to tuberculosis. That so few of those infected suffer in any way from their infection suggests, paradoxically, that man possesses a high degree of resistance to this disease.

We are not in a position to state what proportion of any community is able to withstand infection when first exposed to the tubercle bacillus. All children do not become infected at the same time, when exposed to equal infection from the same source; but few, if any, of them resist for long the repeated infections of a contaminated family environment. Man's natural resistance is not adequate to prevent infection where contact has occurred, nor is this resistance affected by the state of general health. The strong and husky are as liable to infection as the weak and puny. In civilised countries this natural resistance is, however, of such high order that bacilli of first infection invade, multiply, and become fixed in the tissues without causing appreciable disturbance to the infected individual. In a certain proportion of cases (24 per cent.—Macpherson, 1939) the development of the well-known Ghon focus marks the site of first infection, and can be demonstrated during life; but in a larger number of individuals the only evidence of infection is the positive reaction to tuberculin. Resistance at this high level does not, however, seem to be a natural attribute of all mankind, but would seem to be acquired by those races or communities which have had long experience with tuberculosis (Hall, 1935). This heightened "racial" immunity is probably a result of natural selection, as only those persons with some resistance to infection are able to survive the acute forms of disease that occur in a previously uncontaminated community (Borrel, 1920). The "primitive" tuberculosis of young races represents an intermediate degree of acquired natural resistance (Lyle Cummins, 1939).

Progressive Primary Lesions.—In addition to its inability to prevent infection, natural resistance, even at its heightened "civilised" level, is not always enough to prevent disease and death as the direct results of primary infection. Many such results can often be explained by overwhelming or repeated infection, or by some accident of situation of the primary complex. Thus, though death from miliary tuberculosis and meningitis may occur as the direct result of large primary infections, many similar cases are, in fact, the result of secondary infection; and in them well-developed and controlled primary complexes can be demonstrated (Wallgren, 1934; Myers, 1936). The accidental lodgment of bacilli of first invasion near a bronchus may lead to cavitation and, later, bronchogenic spread, and so give the impression of a progressive primary lesion.

Primary infection rarely produces any symptoms or signs of ill-health, even when these are carefully looked for (Israel and Long, 1941). Occasionally a pleural effusion develops with its classical symptoms and physical

signs. This may proceed to empyema and extension, and so lead to serious ill-health and even death (Louria, 1940). When it is considered that the majority of primary foci are laid down immediately beneath the pleura, it is surprising that effusions are not more common. An effusion is, however, strictly speaking, a phenomenon of the secondary period and occurs with, and probably on account of, the development of allergy. Epituberculosis must also be considered as a complication of primary tuberculosis. Though occurring in the allergic period, it is now known that the enlargement of the hilum glands related to the primary focus causes collapse of a main bronchus and atelectasis of the corresponding lobe. It runs a benign course and clears without treatment, but may be responsible for the development of bronchiectasis in some children.

We have reason to think, then, that, as usually acquired, primary tuberculous infection is benign; but accidents of placement leading to extension or to allergic phenomena in the secondary period may give the impression of progressive disease.

Primary or Post-Primary Dissemination.—In some cases a bacteriæmia and more or less generalised dissemination of bacilli occurs during or soon after the development of the primary focus. Great interest in this phenomenon has developed with the concept of a "hæmatogenous type" of tuberculosis. Some observers state that this general dissemination is an invariable accompaniment of primary infection, but find difficulty in proving it. Thus, Pagel states: "There is no primary infection without accompanying or subsequent bacteriæmia" (Kayne, Pagel, and O'Shaughnessy, 1939, p. 91). This statement is modified on the next page when he refers to the "bacteriæmia which is almost invariable in primary infection." He was able to find evidence of "abortive hæmatogenous dissemination in 10 per cent. of 7,000 necropsies in which tuberculosis was an accidental finding." How primary tuberculosis, so typically a lymphatic infection, invariably produces a bacteriæmia is not stated; but we are told that a primary lesion may liquefy and erupt into vessels, or that bacilli may be transmitted from the hilar glands to the junction of the subclavian and jugular veins (*ibid.*, p. 63).

McCordock (see Stewart, *et al.*, 1937) states that in most instances the primary infection is not localised to the lungs and regional lymph nodes, and that isolated tubercles can be found in other organs. He emphasises the fact that this dissemination takes place "during primary infection," and explains it by stating that "mononuclear phagocytes carrying bacilli may pass through the regional lymph nodes to enter the thoracic duct and eventually reach the circulation."

Bacilli entering the venous circulation, whether in phagocytic cells or

as free particles, will be subjected immediately to the filtering action of the pulmonary capillaries, and many of them will again find lodgment in the lungs. These may account for the development of the calcified Simon's foci, which resemble the primary focus so closely both in age and nature. They may also lay the foundations of a later developing hæmatogenous type of pulmonary tuberculosis and the chronic miliary tuberculosis of Hoyle and Vaisey (1937). Bacilli escaping filtration in the pulmonary circulation will enter the general arterial circulation and so plant the field for the subsequent development of tuberculous lesions in the bones, joints, and other organs.

It is not possible to think of any general dissemination that does not produce a grave effect on the health of the individual once allergy has developed. Dissemination should therefore be considered as part of the primary phase; and if it is a general occurrence, the fact that it is so frequently completely controlled serves to demonstrate once more the remarkable degree of resistance to tuberculosis possessed by the " non-immune " human being.

Age.—At one time it was thought that any tuberculous infection occurring in a child under the age of two years was inevitably progressive and fatal (McNeil, 1929). The careful observations and statistics of Miriam Brailey (1936) show that this is not the case. Of white children living in a tuberculous environment in Baltimore and showing a positive tuberculin reaction before reaching the age of two years, 87 per cent. were still alive and well at the age of five years. The mortality of coloured infants under similar conditions was higher, and 31 per cent. of these failed to reach the age of five. It must be remembered in this connection that children exposed to such early infection are thereby exposed to heavy and to repeated infection in most instances. Findings very similar to these are reported by Rosenberg and Kereszturi (1937) for infected children in New York, used as controls for a B.C.G. vaccinated group.

Attention was first drawn to possible dangers of late primary infection by Heimbeck (1928), who found a high incidence of tuberculous disease developing in those nurses undergoing training in the general hospital in Oslo who were found to have had a negative tuberculin reaction when admitted to training. These observations are confused by the striking difference in the infection rate of those nurses in question (50 per cent. positive) and the general population of Oslo city (85 per cent. positive at the age of nine years), a difference leading to the inference that many previously reacting foci had healed, which is extremely unlikely. Since Heimbeck's original observations much attention has been given to the professional hazard run by medical students and nurses whose duty often

takes them into contact with open tuberculosis. Most records show that clinical tuberculosis develops more frequently in the tuberculin-negative medical students or nurses (all of whom develop positive tuberculin reactions) during or soon after their clinical training than in those known to harbour a prior infection (Scheel, 1935; Edwards, 1937; Myers *et al.*, 1937; Geer, 1935).

A recent intensive study of this problem in 3,336 students entering Lund University shows that in spite of a moderately high general rate of infection (80 per cent. tuberculin-positive at fourteen years) the tuberculous morbidity amongst medical students and nurses (11·3 per cent.) was considerably higher than that of other students—1·9 per cent. (Hedvall, 1940). A study of the primary lesions (Malmros and Hedvall, 1940) developing during the course of these observations showed that about one-third (47) of those students becoming infected (151) showed some tuberculous change and that these changes were frequently " rather malignant and progressive." Thirty-five of the forty-seven showing evidence of their infection were medical students or probationer nurses. In only fourteen were typical " primary complexes " demonstrated without other lesions. In another fourteen, " pulmonary tuberculosis " developed without any evidence of a primary lesion The value of these statistics for general application is thrown into considerable doubt when it is admitted (Hedvall, 1940) that sixteen primary infections were acquired by medical students during a course in general pathology, and before there had been any clinical contact with patients. These were probably owing to infection in the post-mortem room—a hazard peculiar to the institution at the time of the survey, and since rectified.

In spite of the uncertainty caused by these and similar studies, it seems that a primary infection acquired under normal conditions, whether in extreme infancy, adolescence, or adult life, is not more malignant than that developing during childhood.

Situation.—Primary tuberculosis may presumably develop in any organ or tissue of the body. Because of their greater exposure, the lungs and the intestines are the most frequently affected. The apices of the lungs are less likely to show the scars of invasion than the remainder of the lung field, and the primary nodule is frequently situated immediately beneath the pleura.

The point of entry of bacilli through the intestinal wall can seldom be found, possibly because any primary lesion developing there heals quickly and completely, or possibly because bacilli are able to penetrate the mucosa without causing any local lesion, and produce their first reaction in the draining lymph glands.

Primary infection of the skin is not unknown (*e.g.*, circumcision tuberculosis). Michelson (1935) has reviewed the literature and reported one case in detail.

It is possible that the tonsil may be a portal of entry of bacilli of first infection more frequently than is supposed, and that cases of cervical adenitis represent a common morbid form of primary tuberculosis.

Something resembling a primary focus in the portal system has been described by Pagel in one of the rare cases of congenital tuberculosis (Kayne, Pagel, and O'Shaughnessy, 1939).

Bacteriology.—Primary tuberculosis may result from infection with either a bovine or a human type of bacillus. The lesions which result are not distinguishable by ordinary pathological examination. Blacklock (1932) found a wider dissemination and a greater tendency to generalisation of the disease when the human bacillus was the primary invader than was the case with the bovine bacillus, and this was true irrespective of the site of primary invasion.

Results of Primary Infection.—The profound changes that take place more or less permanently in all tissues of the body as the result of a primary tuberculous infection are out of all proportion to the insignificance of the reaction of the body to the primary invaders. This allergy (altered reaction to infection) is the property of the tissues, and remains as long as there is any " anatomic " tubercle left in the body. It seems to comprise at least two components, which can with difficulty be separated the one from the other. As a result of a prior infection experimental animals develop an " acquired immunity " which enables them to withstand larger doses of virulent bacilli than their unprotected controls. At the same time they develop a " tissue hypersensitivity " which renders them acutely and even dangerously ill when they are reinfected with a dose of bacilli which leaves their non-immune neighbours completely indifferent for some considerable time. Our knowledge of allergy in the human being depends on our ability to observe and measure the second component in the sensitivity of the skin to tuberculin. This tuberculin sensitivity is a fluctuating affair, waxing and waning under conditions not completely understood, but probably disappearing only in the moribund, or when every tuberculous lesion in the body is healed. Such a state of acquired anergy cannot be of frequent occurrence where infection is widespread, but may be disturbing in mass surveys in districts with a low tuberculosis infection rate (Gass, *et al.*, 1938).

What should one think of the primarily infected human being? Is he " immune " or is he " sensitive "? We have seen how susceptible man is to infection, and yet how resistant to disease. A prior infection seems

to provide a certain immunity to reinfection, so that reinfection tuberculosis is a less easily acquired condition than primary tuberculosis. Where reinfection has occurred, however, disease results and is liable to be progressive; in fact, it is only in allergic soil that tuberculosis becomes a progressive disease. Stewart (1935), from his experience in the Lymanhurst School for infected children, has formed the opinion that not only does primary infection fail to act as a protection in the event of reinfection, but that it is actually detrimental; for by producing chemical changes in the tissues it is the prerequisite for the production of phthisis and other destructive forms of tuberculosis. This opinion was confirmed by a panel of experts, of which he was chairman, which met in Kansas City in May, 1936, and is expressed in his words that " the normal state of the uncontaminated body is superior to the combined immunologic and pathologic post-infection state " (Stewart, *et al.*, 1937).

Treatment.—The majority of human beings reaching adult life have experienced primary tuberculosis which has progressed to healing without symptoms, and so without treatment. Even when a massive infection is discovered in an active form, apparently no known therapeutic measure modifies its course. Treatment is limited to bed-rest while symptoms are present, and to the observance of hygienic and dietary measures to promote optimum health and nutrition (Stewart, 1936). Institutional care is not necessary; and children usually do better at home, provided the source of contagion is removed (Stewart, 1936; Korns, 1934). Macpherson (1939), however, advises that London children of the hospital class should be treated in institutions whenever unhealed primary lesions are found, even in the absence of symptoms. The possibilities, as well as the need, for treating primary tuberculosis are limited. Preventoria, summer camps, and open-air schools supply little of benefit that is not equally and far more economically available in the average tuberculosis-free private home. Children with primary tuberculosis may safely live a normal family and school life, and should not be treated as chronic invalids as long as they are free from symptoms.

Significance of the Healed Primary Complex.—Healed primary complexes as revealed by the presence of calcified nodules in the peripheral lung field and/or calcium in the hilum lymph nodes are a common finding in radiographs of the lungs of healthy persons. There is no reason to believe that these persons are more liable to develop reinfection tuberculosis than those who, though reacting to tuberculin, give no visible evidence of the site of their first infection; nor is there evidence to show that those in whom the primary complex is large and striking are in any great danger. Stewart (1933) states that all children with positive tuberculin reactions, irrespective

of whether or not there is evidence of "childhood tuberculosis,"* are equally liable to develop phthisis. Weintraub (1936), after five years' observation, found that tuberculin-positive children showing evidence of childhood tuberculosis were no more prone to develop lesions of the adult type than those with normal X-ray findings. Zachs (1931), however, found that children who showed evidence of a childhood infection in the lungs developed reinfection tuberculosis twice as frequently as a control group whose prior infection was apparent only as a positive tuberculin reaction. He considers that the discovery of primary nodules, regardless of their size, is of significance as showing a source of infection in close contact with the child, and their liability to reinfection therefrom.

Summary and Conclusions

Though itself a benign condition, primary tuberculosis produces changes in the tissues which set the stage for the malignant and progressive forms of reinfection tuberculosis. Were the immunological changes produced by the first infection of such an order that reinfection of allergic tissue were prevented, tuberculosis would cease to exist as a disease of mankind. But this is not the case; and though reinfection may be resisted, it is not prevented. In this respect, tuberculosis seems to differ from the other great granulomatous disease, syphilis, in which primary infection is invariably progressive and in which reinfection cannot take place while disease, even though it be entirely latent, exists in the body.

The ideal state for the community is that of complete freedom from infection, and efforts to control tuberculosis should be directed towards the eradication of all sources of infection, rather than towards the production of specific immunity. Where, however, infection and the risk of early and repeated reinfection may be expected, some value to the individual may result from an artificially produced infection with non-virulent bacilli, provided time is given for immunity to develop and tissue hypersensitivity to wane before exposure to reinfection is allowed.

The discovery of a primary infection in a child should not condemn it to a life of chronic invalidism, but should lead to the examination of its contacts with the expectation of the discovery of a source of infection, the successful handling of which will protect the child from any consequences of its infection, and possibly save the life and health of many other persons.

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* The term "childhood tuberculosis" is here used, as defined by the National Tuberculosis Association of America, to represent a primary intrathoracic infection.

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THROMBOSIS OF THE SUPERIOR VENA CAVA AND PULMONARY VEINS

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THROMBOSIS of the superior vena cava is an uncommon condition, accounting for not more than 3 per cent. of superior vena caval obstructions, according to Fischer (1904). As recently as 1936 Oschner and Dixon (1936) were able to collect only 120 examples; of these, 29 per cent. arose from external pressure, 23 per cent. from mediastinitis and 36 per cent. from phlebitis. In the latter group luetic, pyogenic or tuberculous infections are acknowledged causes, and acute or chronic rheumatism may account for a few; but when all is said the majority find no adequate explanation.

In a recent paper in this journal Buzzard (1940) reported such a patient who recovered, and whose thrombosis was regarded as part of a migrating thrombo-phlebitis. The present report, supported by necropsy evidence, concerns another instance with thrombosis of pulmonary veins as well.

Case Report.—A. J., a married woman aged thirty-six years, was admitted to the Brompton Hospital in April, 1940. Apart from scarlet fever and measles, she had been healthy until at the age of seventeen she had a single hæmoptysis of about 10 ounces without subsequent disability. She then remained well until the age of twenty-eight, when a hysterectomy was required for protracted menorrhagia and metrorrhagia. A left femoral venous thrombosis complicated the post-operative course A few months later a series of small hæmoptyses lasted for three days, accompanied by fever for a week. She went to a sanatorium for three months, where a scar on her lung was mentioned. During this time she noticed a feeling of fullness in her neck, and subsequently began to suffer from shortness of breath on exertion; but otherwise she remained well and active for about ten months, until March, 1934. Then she had an attack of "bronchitis" with cough and sputum which lasted for three weeks. After this bout dyspnœa increased and she noticed also cyanosis on stooping. In the following years her dyspnœa increased gradually, until in May, 1939, a right pleural effusion was recognised, and her symptoms relieved considerably by its aspiration. Aspirations had to be repeated more and more frequently, but with less and less relief.

When she came under observation she was markedly cyanosed and orthopnœic. The cyanosis was universal in distribution, giving a plethoric appearance with dilated venules over both cheeks. Both jugular veins were distended—the right more than the left. The superficial veins of the arms were all prominent, the right side again larger than the left. Many tortuous veins were seen over the chest and abdomen with their blood flow in a downward direction. Dilated veins were absent from the lower limbs and there was no œdema.

The pulse was regular and its rate between 80 and 90; the heart was normal in position and there was no clinical evidence of enlargement. The sounds were normal and no murmurs were present. The blood pressure was 128 systolic/84 diastolic in both arms. The circulation time with decholin (arm to tongue time) was twenty-five seconds, the normal reading being nine to eighteen seconds. The venous pressure in the median basilic vein was 13 cm. of water above the level of the manubrium sterni; in the leg the level did not reach that of the manubrium. An electrocardiogram showed right axis deviation with inversion of the "T" waves in all leads. The respiratory rate was moderately raised, and she had the signs of an effusion on the right side. Clubbing of the fingers was absent. The abdomen and nervous system were normal, and the urine also. She was afebrile with no wasting and no clinical reason for thinking that she might have an obscure focus of sepsis. There were no palpable lymphatic glands. A blood count revealed: R.B.C. 5,950,000 per cm.; Hb 112; W.B.C. 11,600 per cm., with a normal differential count. The Wassermann reaction in the blood serum was negative.

A radiograph of the chest (Fig. 1) confirmed the presence of an effusion, and the absence of cardiac enlargement or any abnormality of the aorta. The hilar shadows on both sides showed changes consistent with vascular engorgement; but in addition on the left side, reaching from the aortic arch below the left pulmonary artery, there was a dense opacity with a sharp outer margin running obliquely upwards and inwards. In a shallow right oblique position (Fig. 2) after diagnostic aspiration and air replacement of the pleural fluid this area showed as a circumscribed shadow with a rounded convex outer border, tailing above into the edge previously described. The whole appearance closely simulated a mass of moderate density situated in the hilum. A somewhat similar appearance could be made out on the right side, but not so distinctly.

The fluid in the right chest reaccumulated rapidly and was removed at intervals over two months; but her dyspnœa became steadily less amenable to such relief, and her general condition slowly deteriorated. It was found that oxygen made no difference to her cyanosis. The fluid was straw-coloured and contained lymphocytes; no bacilli or malignant cells were seen. By July she had developed a tic-tac rhythm and her blood pressure had fallen to 100 systolic/80 diastolic. Her circulation rate at this time was eleven seconds in the right and eighteen seconds in the left arm with ether (arm to lung capillaries), the normal being ten seconds; and thirty seconds in the right and thirty-four seconds in the left arm with decholin. The urine showed a trace of albumen, and the output had fallen to 10 to 15 ounces daily; salyrgan, urea and ammonium chloride increased the output to 30 to 40 ounces daily.

This patient thus presented the classic features of obstruction of the superior vena cava with severe dilatation of its tributaries, well-marked collateral circulation with the blood flow towards the inferior caval system, and a venous pressure raised in the arm, but not in the leg. The delayed circulation time with decholin supported this evidence, as also did the absence of any evidence of intrinsic heart disease, chronic pericarditis or chronic pulmonary disease. The cause of the obstruction was uncertain. Aneurysm was already excluded, and bronchial neoplasm seemed unlikely, particularly on account of the length of history. At this point bronchoscopy was desirable, but was not possible because of the severity of her breathlessness. The likeliest causes of the obstruction appeared to be thrombosis, pressure by a localised tumour, or constriction by an adhesion. In view of her steady deterioration and the possibility that an obstructing factor might be removed or the mediastinum decompressed, an exploratory thoracotomy was undertaken by Mr. Price Thomas.

Under local anæsthesia supplemented by cyclopropane a curved incision was made anteriorly and a flap including the right breast was reflected upwards. A transpleural, rather than extrapleural, route had then to be followed because greatly thickened pleura could not be reflected from the superior mediastinum. The pleural cavity contained several ounces of straw-coloured fluid in loculi; the upper lobe of the lung was aerated, but adherent to the chest wall over the dome; the lower lobe was completely collapsed. Grossly thickened parietal pleura was reflected from the superior mediastinum to expose the pericardium, which appeared to be

pulsating normally. The superior vena cava just above its entrance into the right auricle consisted of a firm non-pulsatile cord one-third of an inch in diameter; it was traced upwards and freed from the adjacent thickened and tough tissues, but normal vein could not be reached. Freeing of the vena cava had no effect upon its size. Further dissection could not be undertaken safely, and the pleural cavity was closed after inserting a malecot catheter for drainage.

The immediate post-operative course was uneventful, and, if anything, her cyanosis, breathlessness and venous dilatation appeared less than before. But improvement was short-lived, for her condition gradually deteriorated, and she died about three weeks later.

Necropsy.—At necropsy the upper lobe of the right lung was aerated, but the lower and middle lobes were atelectatic. On the right side the visceral and parietal pleuræ were considerably thickened and a grossly loculated hydro-pneumothorax was present. On the left side the lung and pleura were normal, excepting that the lingula process existed as a separate lobe. No tuberculous foci were detected, although a careful search was made. The heart was not enlarged, but the muscle was pale and flabby. There was no valvular abnormality, but a small patency of the inter-auricular septum ($\frac{1}{2}$ inch) was present. The distal $\frac{1}{2}$ inch of the superior vena cava and proximal $\frac{1}{2}$ inch of both right and left innominate veins were represented by small, firm, fibrous cords $\frac{1}{4}$ to $\frac{1}{8}$ inch in diameter, with complete occlusion of the lumen. The orifice of the azygos vein was considerably reduced in size and would only just admit a large probe. Both right pulmonary veins and the left upper pulmonary vein were thrombosed into solid fibrous cords, each little more than $\frac{1}{8}$ inch across; their auricular orifices appeared as small dimples of scar tissue flush with the auricular wall. The left lower pulmonary vein was patent, normal in size, and entered the left lower lobe. An abnormal vein about $\frac{1}{2}$ inch in diameter extended upwards from the hilum of the left upper and middle lobes to the left and in front of the aortic arch joining the left innominate vein (Figs. 3*a* and 3*b*). Apart from an enlarged and somewhat congested liver, there was no other abnormality.

Non-viscous neo-hydriol was injected into the abnormal vessel entering the left upper and middle lobes, and subsequently radiography showed opaque material in both these lobes. The azygos vein, superior vena cava, innominate veins and the abnormal venous channel to the left lung were then plugged with cotton-wool impregnated with barium and further radiograms taken (Figs. 4*a* and 4*b*).

Histological examination of the thrombosed vessels showed complete occlusion by fibrous tissue without any re-canalisation. No inflammatory cells were found in the thrombus or in the walls of the veins, and evidence of tuberculosis, syphilis or neoplasm was lacking.

Discussion and Summary.

In this remarkable patient organised thrombosis of the superior vena cava is associated with the same condition in three out of four pulmonary veins, a combination which does not seem to have been recorded previously.

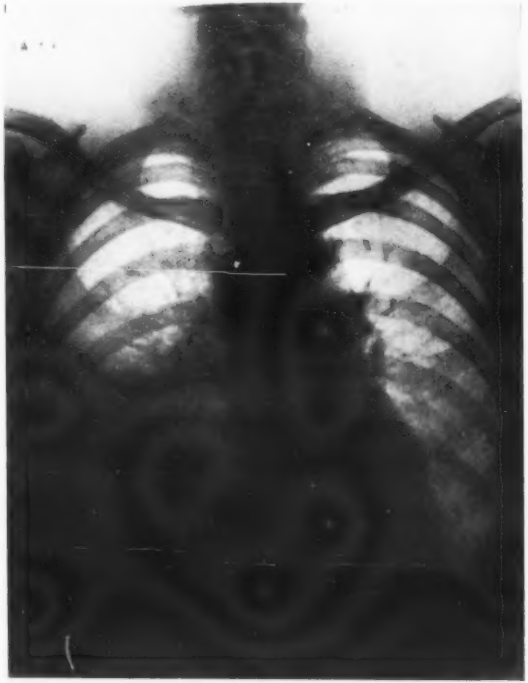

FIG. 1.—POSTERO-ANTERIOR RADIOGRAPH OF CHEST SHOWING RIGHT HYDROTHORAX AND OPACITIES IN BOTH HILAR REGIONS FROM THROMBOSIS OF PULMONARY VEINS.

That on the left side has a curved outer border extending upwards, representing the abnormal venous channel from the left lung to the left innominate vein (necropsy confirmation).

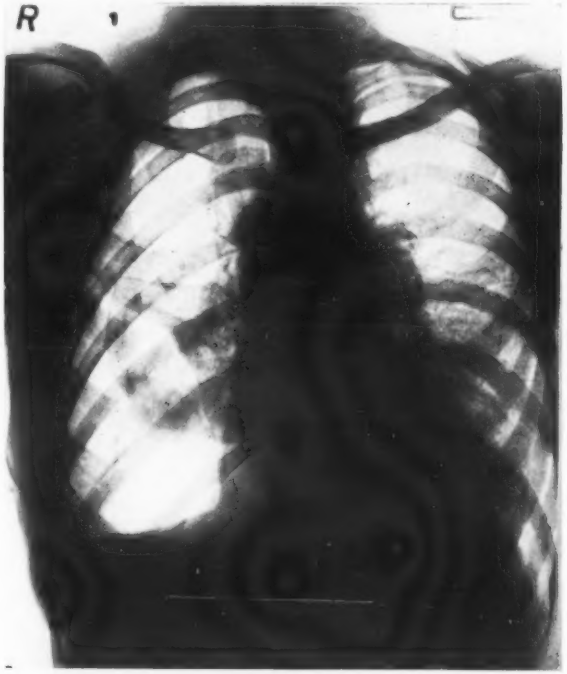

FIG. 2.—SHALLOW RIGHT OBLIQUE RADIOGRAPH OF CHEST AFTER ASPIRATION AND AIR REPLACEMENT OF PLEURAL EFFUSION, SHOWING RIGHT HYDRO-PNEUMOTHORAX AND LEFT HILAR SHADOW PARTLY SEPARATED FROM THE ABNORMAL VESSEL SEEN IN FIG. 1.

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PLATE XVII

FIG. 3*a*.—NECROPSY SPECIMEN, SHOWING THROMBOSED PORTION OF SUPERIOR VENA CAVA AND INNOMINATE VEINS AS WELL AS THE ABNORMAL VENOUS COMMUNICATION BETWEEN LEFT LUNG AND LEFT INNOMINATE VEIN.

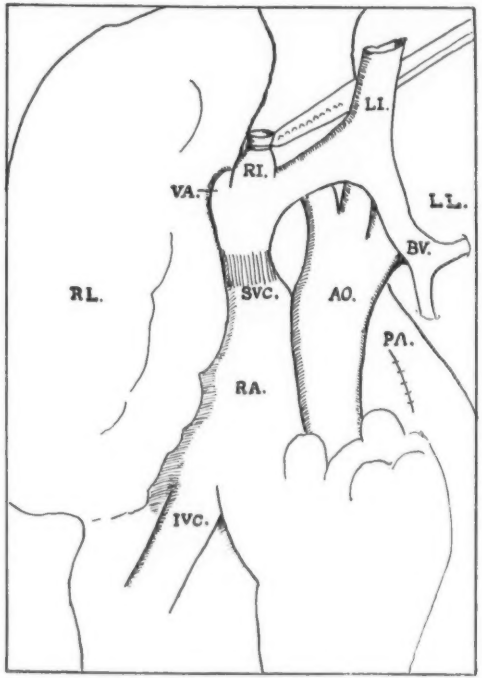

FIG. 3*b*.—DIAGRAMMATIC REPRESENTATION OF FIG. 3*a*.

RI., Right innominate vein; LI., left innominate vein; BV., abnormal venous channel; VA., azygos vein; SVC., superior vena cava; RA., right auricle; IVC., inferior vena cava; AO., aorta; PA., pulmonary artery; RL., right lung; LL., left lung.

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PLATE XVIII

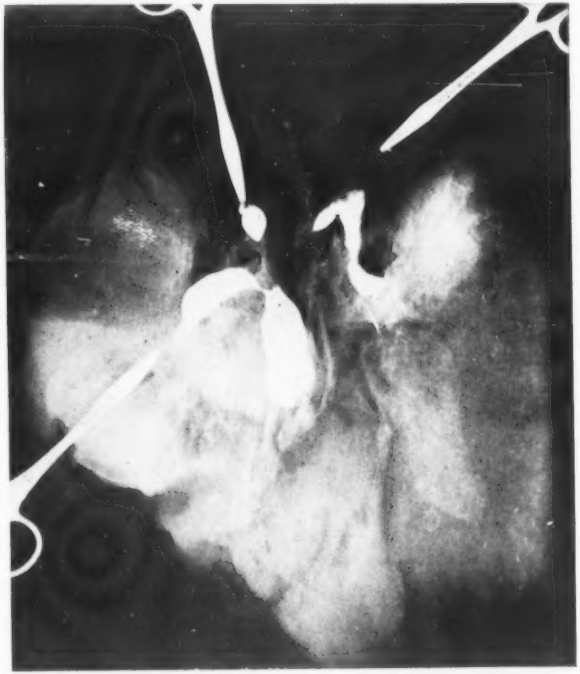

FIG. 4*a*.—RADIOGRAM OF THE LUNGS AFTER INJECTION OF NON-VISCOUS NEO-HYDRIOL INTO THE ABNORMAL VENOUS CHANNEL AND PLUGGING THE VEINS WITH COTTON-WOOL IMPREGNATED WITH BARIUM, SHOWING THE THROMBOSED PORTION OF THE SUPERIOR VENA CAVA AND INNOMINATE VEINS AND THE ABNORMAL VEIN ENTERING THE LEFT LUNG.

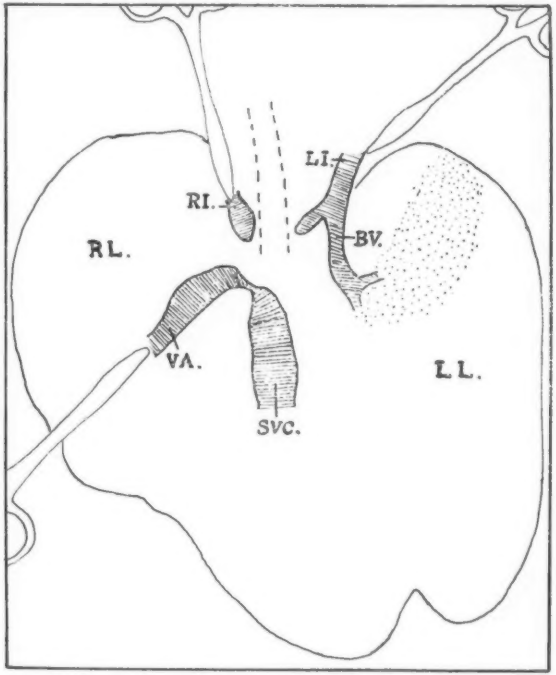

FIG. 4*b*. —DIAGRAMMATIC REPRESENTATION OF FIG. 4*a*.

RI., Right innominate vein; LI., left innominate vein; BV., Abnormal venous channel; VA., vena azygos; SVC., superior vena cava; RL., right lung; LL., left lung. The stippled area represents the portion of the left upper lobe filled with neo-hydriol.

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The earliest incident which might be taken as pointing to the onset was the single hæmoptysis at the age of seventeen. Nothing more happened for twelve years, when a femoral thrombosis complicating a hysterectomy performed for uterine bleeding suggested that pelvic thrombosis might have been under way at that time. Within a few months of this, hæmoptysis recurred, and it was from this time onwards that the features pointed progressively to the extensive venous occlusions, though in a most gradual way until within a year of her death. It is clear that during this last year a terminal stage had already been reached because by that time there was an extensive collateral circulation, a massive and recurrent pleural effusion, and fibrous transformation of old-standing thrombi. The surprising thing is that such slight symptoms earlier on most probably indicated the original thromboses. Considering how complete these were eventually, it is difficult to escape the conclusion that the process had been slowly progressive. But the ordinary causes of venous thrombosis were not found and the explanation in this patient remains a mystery.

Reviewing the clinical features, it does not seem possible to select any that pointed to the pulmonary venous thrombosis during life, excepting the two hæmoptyses and the peculiar radiological picture at the hilar regions. Although it is not certain, it is tempting to regard the abnormal shadowing here on both sides as due to the actual thrombosed veins, for no other explanation could be found at necropsy. On the left side the evidence is strong, for the sharply defined curved outer edge extends upwards and corresponds in position with the abnormal communicating vein. The pleural effusion cannot be regarded as special evidence of the pulmonary venous changes because the thrombosis in the superior vena cava involved the azygos, and this, in any event, would have led to hydrothorax.

It is difficult to understand the course of the venous return from the right lung, as no evidence of abnormal communications could be found. It is possible that abnormal channels may have been present between that portion of the right lung adherent to the chest wall and the intercostal veins; such a collateral development might well have contributed to the greater venous distension over the right chest wall, for it would produce a venous shunt between systemic and pulmonary circulations. The abnormal venous channel issuing from the left upper and middle lobes and entering the left innominate vein was in all probability a very much enlarged bronchial vein, for the latter not infrequently enter the innominate.

The relatively slight rise of venous pressure in the arms agrees with the well-developed anastomotic channels, as it is known experimentally that chronicity lowers the height of the venous pressure. The original circulatory rate with decholin was not much raised, surprising in view of the com-

plete obstruction; the added rise just prior to thoracotomy was probably attributable to some degree of cardiac failure, evidenced as well by a tic-tac rhythm and a fall in blood pressure.

There are other difficulties in the interpretation of the circulation times in this patient, particularly the normal ether relative to the abnormal decholin time. One explanation may be that blood from the arm entering the left innominate vein passed through the abnormal venous channel to the lung. The similarity in the readings of each arm could only be explained by an excellent communication between the right and left innominate veins in the neck or upper thorax. This explanation would imply a flow of blood in the abnormal vessel from the innominate vein to the lung, but in this case it is still difficult to understand the venous return from the left upper and middle lobes, as there were no other venous channels leaving by the hilum, and no evidence radiologically of any communication between the vascular systems of the upper and lower lobes after the injection of opaque material into the abnormal venous channel.

My thanks are due to Dr. A. Hope Gosse and Mr. Price Thomas for permission to publish this case.

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OBSERVATIONS ON THE COMPLEMENT FIXATION TEST IN TUBERCULOSIS

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I

THE complement fixation test plays only a small part in the diagnosis of tuberculosis. The majority of physicians do not rely on this test in doubtful cases, and in England particularly it is unusual even to use the test when tubercle bacilli cannot be found in the sputum, or for the diagnosis of inflammatory effusions.

A number of attempts have been made to establish a serum test in tuberculosis for use as a routine method. Many immunologists have tried to elaborate such a test for tuberculosis by the production of suitable

antigens and by using a special technic for the complement fixation test. Besredka's test[1] deserves first mention. He recommends as an antigen a fine emulsion of killed tubercle bacilli which are young, and therefore have no wax coat, cultured on an egg-yolk medium; he makes use of a modification of the Calmette-Massol method. Boquet and Nègre[2] employ as antigen a methyl alcoholic extract of tubercle bacilli treated previously with acetone, and they utilise the Calmette-Massol method as well. Further, A. v. Wassermann[3] devoted the last years of his life to investigations on this subject; he used as an antigen tubercle bacilli which had been extracted with tetraline, and brought the residues into an emulsion with lecithin. G. Blumenthal[4] obtained a useful antigen by acetone extraction, ether treatment and solution of the antigenic constituents in 0·5 per cent. phenol. The antigen of Witebsky, Klingenstein and Kuhn[5] (pyridine extraction of tubercle bacilli boiled with hot alcohol, freed of the constituents soluble in cold alcohol) is frequently used.

Many investigations have been made in America on the antigenic action of various fractions of the tubercle bacillus and the usefulness of the test. I need only mention two important papers of Pinner[6] and of Wadsworth[7] and his collaborators. Pinner demonstrated that the alcohol soluble, acetone insoluble substances of the tubercle bacillus play by far the most important part as antigens in the complement fixation test. Wadsworth, F. Maltana and E. Maltana undertook extensive investigations to establish a useful routine serum test; they selected preparations from virulent human and bovine strains of tubercle bacilli as follows: (1) dialysed distilled water extract antigen; (2) purified and concentrated aqueous extract antigen; (3) purified and concentrated culture filtrate antigen. They give a detailed report of all their cases.

In this country Sellers and Ramsbottom[12] concluded that the test is not reliable, too little attention having been paid to so-called non-specific reactions. Coulthard,[8] however, gave a favourable report about the complement fixation test in tuberculosis. According to him suspensions of killed tubercle bacilli in saline are reliable, stable and sensitive antigens; in general they give the most marked reactions. Next in value comes the alcoholic extract obtained from cultures of tubercle bacilli which have been previously treated with acetone. With lipoid antigen often stronger complement fixation is obtained than with suspensions of bacilli in saline; but certain tuberculous sera which show a reaction with the latter fail to fix complement in the presence of the lipoid extract. He remarked that the reaction appears to be highly specific.

II

As a collaborator of A. v. Wassermann I took part in his research work, and after his death carried on further investigations[9] in collaboration with C. Neuberg. We used an antigen in which the splitting up of the tubercle bacilli and the solution of its specific constituents was carried out with 10 per cent. sodium benzoate, using the principle of hydrotropia. The results were not altogether satisfactory. We then began systematic investigations, splitting the tubercle bacilli in single fractions, and comparing the antigenic effect of the constituents in the complement fixation test. In this way we explored:

1. The wax and fat fraction obtained by the use of wax dissolving mediums (acetone, tetraline, epichlorhydrine-chloropropylenoxide, chloroform, toluol, and other solvents used in the lac and varnish industry).

2. The lipoid fraction which is obtained from tubercle bacilli by alcohol extraction after the removal of all wax and fat contents.

3. The protein fraction isolated by different methods. (Compare Felix Klopstock and Carlo Cattaneo, " Antigenic Effect and Chemical Structure of the Tubercle Bacillus," *Zschr. f. Immun. Forsch.*, 1935, vol. 84, p. 34.)

4. The carbohydrate fraction. (Compare *ibidem.*)

These investigations demonstrated the superiority of the lipoid extracts beyond doubt. The ethylalcohol extracts were the best. Methylalcohol extracts endanger the specificity of the antigen. The splitting up of the lipoid fraction into further constituents and the use of a phosphatide did not improve the results of the complement fixation test. The performance of the test with chemically pure products did not further the intensity or specificity of the reaction.

We therefore recommended a tubercle bacilli antigen obtained by ethylalcoholic extraction of the bacilli after the removal of the wax and fat contents by epichlorhydrine; the latter removes all these constituents effectively and abolishes the acid-fastness of the bacilli. During newer investigations it was found that toluol-extraction of the wax ingredients and alcoholic extraction of the remaining substances also yielded very good antigens. The extraction with toluol must be continued till the acid-fastness is completely abolished. Frequent shaking during the extraction may be recommended.

The efficiency of such extracts can be increased by minute quantities of lecithin or wax substances. For each antigen the necessary amount must first be titrated in a preliminary test. Lecithin and wax substances act as sensitisers; they produce the optimal state of dispersion for the reaction, but they are without influence on the affinity between antigen and antibodies.

The addition of a small amount of carbolic acid (0·5 phenol) amplifies the action of the lipoid antigen. Sachs, Sollazo,[13] and Dreyfuss[14] showed the ability of carbolic acid to intensify the effect of specific and non-specific extracts. Blumenthal demonstrated this capacity for tuberculous antigens also. Our investigations confirmed these findings, but the addition of lecithin or wax substances seemed superior as to the specificity and sensitivity of the reaction.

III

In the complement fixation test in tuberculosis the reaction between the specific constituents of the extracts and the antibodies of the serum does not produce the inactivity of complement with the same regularity and intensity as in the W.R. for the serodiagnosis of lues. Practically any technique for the complement fixation test can be employed: it can be used either with a fixed dose of complement, and the titer of amboceptor can be defined in the preliminary test—or the complement dose can be titrated in the preliminary test—or the method of Calmette-Massol can be used where titration of the complement is transferred to the main test.

The following points need to be kept in mind:

1. The reaction should be done with inactive serum. The use of active serum gives a greater extension of the reaction, involving, however, the danger of a non-specific reaction. Different intervals between taking the blood and the investigations in the laboratory influence the result.

2. The serum must be examined, not only in one dilution (1 : 5), but also in the dilution 1 : 2·5 and 1 : 10. The dilution 1 : 5 is not always optimal; stronger and also weaker concentrations may yield a more distinct reaction. The optimal ratio of antigen and antibody fluctuates much more in the tuberculous reaction than in the lues reaction.

3. Strongly hæmolytic sera and sera yielding even a trace of auto-inhibition must be excluded from the examination.

4. The test must be employed with several extracts. Extracts produced by the same methods do not always agree. The extracts are best used in two doses, in three-quarters of the minimal solvent dose and in about half this dose. Consequently stronger extract-doses must be applied than are usual in other complement fixation tests.

5. In all tests a lues antigen must be used besides the tuberculous extracts. All cases with a positive W.R. which yield at the same time a reaction with tubercle bacilli extracts must be regarded as under suspicion. Syphilitic blood can show reactions with all antigens containing lipoids and thus counterfeit tuberculous antibodies. Even the titration of such sera with the different extracts does not give reliable results.

6. The concentration of complement must be weaker than is usual in the W.R. The intensity of the reaction can be improved by the use of a complement concentration of 1 : 15 or 1 : 20.

7. The incubation period may be lengthened to about one hour, and cooling then allowed for half an hour before the addition of the hæmolytic system.

8. Serological tests in tuberculosis must be performed only by persons familiar with the performance and interpretation of serological tests.

These recommendations are made to prevent the danger of non-specific reactions.

IV

Estimation of the results of serological tests in tuberculosis rests primarily upon animal experiments. Besredka reported in 1914 that in guinea-pigs antibodies can be identified on the fourth day after infection at a time when no anatomical lesions can be observed with the naked eye, and that the antibodies may disappear during the course of the disease, specially shortly before death. In my own experience with tuberculous guinea-pigs the earliest positive reaction was found on the ninth day after infection. A strongly positive reaction appears by the end of the second or in the third week. The serological reaction generally precedes the intracutaneous reaction. The concentration of antibodies fluctuates; a uniform level does not exist, and they regularly disappear in cachectic phases of the disease.

Animals inoculated with extracts from tubercle bacilli also develop a positive reaction. Normal rabbits especially exhibit a strong reaction after intravenous injections of our tubercle bacilli extracts prepared by evaporation and solution of the residues in normal saline. This result is important for the belief that a positive reaction is not necessarily associated with " active tuberculosis " or with the presence of living tubercle bacilli. Clinical and biological activity are not the same thing. The tuberculin test demonstrates that at some time a struggle has taken place between the organism and the contagious agent; the serum reaction, on the other hand, indicates that the reaction of the organism against living or dead tubercle bacilli is still present. But a positive serum test does not imply that there is clinically active tuberculosis.

The results of the serum tests in human tuberculosis vary with the different forms of tuberculosis. The results of Wadsworth and his collaborators in their last series of examinations may be reviewed, because they are based on their experience with a great number of patients (1,002 cases) and because the investigations appear to have been carried out with particular care. The 1,002 cases include 688 in which a diagnosis of tuberculosis

was made and a control group of 314 cases in which no signs of tuberculosis were observed clinically. In the former group fixation of complement was obtained with tubercle antigens in 84·9 per cent. There were, however, a number of well-advanced cases with tubercle bacilli in the sputum and certain incipient cases with a low grade of activity in which no reactions were obtained. Fixation occurred in only a comparatively small proportion of the sera from cases diagnosed as inactive or apparently cured. In the group of non-tuberculous cases positive reactions were obtained in 14 per cent. This percentage includes those cases reacting to the complement fixation test for syphilis.

My own results depend upon investigations over some ten years, but they include results using different methods. In tuberculosis of the lungs an average of 80 per cent. of the cases were positive. The percentage is much lower in early disease. The earliest tuberculous changes rarely yield a positive reaction. In the second stage of the disease the percentage increases to 90 per cent. In the third stage with the occurrence of cachexia negative results are frequently obtained. In childhood the results are less satisfactory than in adults. Acute disseminations of tuberculosis are usually not provable by the test. The test is important for the proof of tuberculous pleurisy.

In surgical tuberculosis the results are less encouraging. My examinations gave only 55 per cent. positive tests. Isolated bone and joint tuberculosis frequently gives a negative result. Urogenital tuberculosis, on the other hand, gives a positive test in a high percentage of the cases, approaching that of the second stage of tuberculosis of the lungs.

In skin tuberculosis nearly 50 per cent. positive results are obtained. It is interesting that two diseases which are not yet proved as tuberculous frequently yield a positive test, lupus erythematosus and granuloma annulare.

From the examination of exudates and articular effusions, and to a much less degree of C.S.F., the complement fixation test may yield support in diagnosis. The test may clarify the tuberculous nature of an inflammation which cannot be proved by any other method of examination. The high fibrin content of effusions requires the test to be performed with special caution and care, for otherwise inhibition of the complement activity by the fibrin content of the fluid will be interpreted as a positive reaction. I recommend dilutions of 1 : 5, 1 : 10, and 1 : 20 for such inflammatory effusions. With this technique effusions of tuberculous pleurisy and peritonitis react positively in nearly 60 per cent. of cases. This is a satisfactory result considering that microscopic examination and cultural investigation are so often in vain. Tuberculous joint effusions give a negative result in two-thirds of the cases. Cerobrospinal fluid nearly

always gives a negative result in tuberculous disease of the central nervous system or of the meninges.

One must always bear in mind that the results depend not only on the production of antibodies, but also on the presence of antigen fixing the antibodies in the circulating blood or in the inflammatory effusions. A negative result does not always indicate that no antibodies have been formed; it may also indicate that the antibodies have already been fixed by antigen in the body.

The specificity of the reaction is reliable with the following qualifications: As already mentioned, W.R. positive cases must be excluded from serodiagnosis, because syphilitic blood can react with all lipoid extracts. The same applies to leprosy. Further, it is advisable not to apply the serum test in recent cases of diphtheria, as the lipoid of the tubercle bacillus and the lipoid of the diphtheria bacillus have a chemical or physico-chemical relation. The necessity of excluding strongly hæmolytic sera or those which show the slightest sign of auto-inhibition has already been emphasised. With these various precautions the complement fixation test demonstrates reliably the occurrence of specific antibodies against the tubercle bacillus.

During the work I tried to differentiate human and bovine tuberculosis by means of the test. Extracts from human and bovine strains of tubercle bacilli were made by the same method and applied to cases of human tuberculosis and tuberculosis in cattle. Sera from tuberculous cattle nearly always gave a strongly positive reaction, and extracts of bovine bacilli seemed superior to those of human bacilli. But they gave somewhat better results, not only in bovine tuberculosis, but also in human tuberculosis, and seemed to have higher contents of specific constituents. Also, examination of fractions other than lipoid extracts gave no reliable results, though recently successes have been claimed in the serological distinction of human and bovine tuberculosis by means of protein antigens of the bacilli (Schäfer,[10] K. Meyer[11]).

The quest of obtaining a serum test in tuberculosis of the same reliability as the W.R. in syphilis has not been fulfilled in my investigations Twenty per cent. of cases with tuberculosis of the lungs and at least 40 per cent. of those with tuberculosis of other organs cannot be proved by the serum test, and a positive test does not permit a clinical diagnosis of active tuberculosis. Nevertheless, the test seems to be a valuable supplement to other examinations. It can secure a diagnosis of "tuberculosis" in a certain percentage of manifest or suspected diseases of the lung without bacilli in the sputum, or of diseases of bones, joints, and skin, and the cause of inflammatory effusions can be clarified in many cases.

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A. STANLEY GRIFFITH

AN APPRECIATION

THE death of Dr. A. Stanley Griffith means a very real loss. Of him it can be said that the whole nation is his debtor.

He graduated from Manchester in 1897, and early in his career began his life's work in the investigation of problems in tuberculosis.

When in 1901 Koch made his unfortunate pronouncements regarding the types of tubercle bacilli found in human tuberculosis, and a Royal Commission was formed to investigate *inter alia* the types of tubercle bacilli, Griffith was selected as one of the investigators. From that time a steady flow of carefully recorded work has been forthcoming from his pen. It would be impossible in a short statement to give any indication of the volume of work, but a brief review shows that he has touched on every aspect of the subject. His work on lupus, his study of B.C.G. in attempted immunisation of calves, his studies on tubercle bacillæmia, his researches on bone and joint tuberculosis, etc., each a finished article, all testify to the accuracy of his work. But Griffith was no lone worker. He welcomed co-operation. It is now twenty years since the writer wrote him detailing a scheme he had in mind for investigations in pulmonary tuberculosis. Griffith's reply was one of advice, help and an offer to co-operate. Thus began an association which was to lead to many joint investigations. His laboratory at Cambridge was unpretentious, but it became the centre to which would come any doubtful acid-fast bacillus, for his was the court of appeal as to the type of any doubtful virus of tuberculosis.

Griffith always insisted that the bovine type bacillus was at least as virulent to the human subject—if not more so—than the human type. He was right.

Griffith was interested, too, in the clinical side of tuberculosis. He came to Scotland on various occasions. He wished to see the clinical states of those cases of pulmonary tuberculosis from whom we had isolated bovine type tubercle bacilli. His inquiry into the history, the possible source of infection, the various manifestations of disease, the family, and the possibility of spread showed the grasp he had of the whole subject and its implications. He enjoyed these visits. He was almost on holiday. He visited farms, visited Glenlomond Sanatorium, East Fortune Sanatorium, and Lochmaben Sanatorium. He had inquiries to make at them all. His last visit to Scotland took us to Aberdeen. Griffith held to the view that the bovine type bacillus would be more readily found in a rural community,

especially where there were many small farms or crofts. Such an investigation was carried out in collaboration with Dr. John Smith, Aberdeen, and they published* a review of the types of tubercle bacilli in pulmonary tuberculosis in North-East Scotland. They found 9·1 per cent. bovine type cases in rural Aberdeen and 4·4 per cent. (13 out of 294 cases) in Aberdeen city (7 of the 13 were from the county).

Griffith had an international reputation, and this was based on the accuracy of his work. Nothing ever went from him which he had not carefully checked. He had friendly contacts in Paris, in Berlin, in Vienna and in Copenhagen. In 1932 he was invited to Berlin to the celebration of the fiftieth anniversary of the discovery of the bacillus of Koch. The writer accompanied him, and it is pleasant to record that German bacteriologists regarded Griffith as the one man who had carried forward the work of Koch.

Tributes were paid to Griffith's work at this celebration, over which Professor Pfeiffer presided. A very modest gentleman, retiring, and one who preferred to work rather than to speak, he was persuaded to open the discussions on tuberculosis at the Centenary Meeting of the B.M.A. in 1932, when he gave a masterly review bringing our knowledge of the bacteriology of tuberculosis up to date from the period when Koch died.

Two years ago he began to fail in health, and it was obvious that he would not be spared for long; but he remained at work, collecting all the facts he could of his life's work, and his latest publications show the same careful work, each line worded correctly, and nothing but what he knew therein.

Dr. Griffith leaves a widow and one son, still at school The writer will miss Dr. Griffith very much, but will have the recollection of pleasant visits to Cambridge in the company of Dr. and Mrs. Griffith and Dr. Fred Griffith. The brothers were great Englishmen.

W. T. M.

* *Lancet*, March 26, 1938.

MEETINGS OF SOCIETIES

JOINT TUBERCULOSIS COUNCIL

A MEETING of the Council was held at the London School of Hygiene and Tropical Medicine, Keppel Street, Gower Street, London, W.C.1, on Saturday, May 24, 1941.

Present.—Dr. G. Lissant Cox, Dr. J. C. Gilchrist, Dr. F. R. G. Heaf, Dr. G. T. Herbert, Dr. L. E. Houghton, Dr. G. Jessel, Dr. S. Vere Pearson, Dr. D. A. Powell, Mr. J. E. H. Roberts, Dr. N. Lloyd Rusby, Dr. Norman Smith, Dr. H. G. Trayer, Professor W. H. Tytler, Dr. E. Ward, Dr. E. Rohan Williams, Dr. R. A. Young and the Hon. Secretary.

1. *Minutes.*—The minutes of the last meeting were approved.

2. *Correspondence.*—Letters were read from (*a*) Dr. James Watt resigning his convenership of the Tuberculosis in War-Time Committee; (*b*) from the Honorary Treasurer pointing out that the North-Eastern Tuberculosis Society had been disbanded for the period of the war.

It was *Resolved* that Dr. Dickenson, their representative, should be invited to attend meetings of the Council despite the cessation of activities of the North-Eastern Society.

Apologies for absence were received from Professor Platt; Sir Wilson Jameson; Drs. W. T. Munro, D. P. Sutherland, N. Tattersall, C. O. Hawthorne and Peter Edwards.

3. *Motion by Dr. Heaf.*—Consideration was given to a motion in the name of Dr. Heaf: "That in order to expedite procedure and increase the efficiency of the work of the Council the present method of forming *ad hoc* committees be discontinued."

After some discussion the motion was lost by seven votes to five.

The Council proceeded then to discuss the second motion of the paper in Dr. Heaf's name: "That small permanent committees be formed with power to co-opt members or non-members specially concerned to report on any subject primarily connected with the branch of medicine or allied sciences on which they have been made the Council's recognised authority."

It was agreed, however, to postpone the discussion of the motion and to incorporate a fresh resolution governing the powers of committees and conveners of committees when the new committees are being set up. (See below.)

4. *Committees for* 1941.—Before proceeding to the appointment of new committees the council *Resolved* "That conveners of present or future

committees be empowered to co-opt on their committees members of the Council or outside members."

This decision was arrived at by a majority of three—eight votes to five.

It was *Resolved* also that the Honorary Secretary should write to the Society of Medical Officers of Health asking whether Sir Wilson Jameson still remains a member of the Joint Tuberculosis Council representing that Society.

It was decided that the words " its indications and scope " be eliminated from the terms of reference of the committee appointed to deal with the " Major Surgical Treatment of Pulmonary Tuberculosis."

Dr. Heaf (Convener of the Post-Graduate Committee) reported that there would be a post-graduate class held at Harefield Sanatorium during the summer and that the fee would be two guineas.

The following committees with conveners were appointed:

Care and Employment : Drs. F. R. G. Heaf and the Honorary Secretary (Joint Conveners), Peter Edwards, C. O. Hawthorne, G. Jessel and D. P. Sutherland.

Milk : Drs. C. O. Hawthorne (Convener), G. Lissant Cox, Sir Wilson Jameson, W. T. Munro and D. P. Sutherland.

Nursing : Drs. H. G. Trayer (Convener), G. Lissant Cox, Peter Edwards, J. C. Gilchrist, F. R. G. Heaf, James Watt and Mr. J. E. H. Roberts.

Post-Graduate : Dr. F. R. G. Heaf (Convener).

Tuberculosis in War-Time : Drs. N. Lloyd Rusby (Convener), James Watt, G. Lissant Cox, J. C. Gilchrist, N. Tattersall, D. P. Sutherland, E. Ward and F. R. G. Heaf.

Major Surgical Treatment of Pulmonary Tuberculosis : Mr. J. E. H. Roberts (Convener), Drs. Peter Edwards, James Watt and R. A. Young.

Welsh Report : Drs. D. A. Powell (Convener), and G. Lissant Cox.

The Institutional Treatment of Children suffering from Pulmonary Tuberculosis : Drs. Peter Edwards (Convener), W. T. Munro, D. P. Sutherland and N. Tattersall.

Definition of Terms : Drs. F. R. G. Heaf (Convener); Peter Edwards, G. Jessel, D. P. Sutherland, James Watt and N. Lloyd Rusby.

Co-operation between Tuberculosis Organisations : The Chairman (Convener), Sir Wilson Jameson, Dr. E. Ward, the Honorary Treasurer and Honorary Secretary.

5. *Care and Employment Report.*—Prior to the consideration of this report (previously circulated) Mr. Roberts raised the question as to the methods of

the Council in securing adequate publicity for the reports and communications of the Council. He stated that there were still many physicians and surgeons interested in tuberculosis who did not know anything of the work of the Council, or even of its existence. He moved that: "This Council ask suitable bodies to grant subsidies for the free distribution of reprint and reports." This was agreed to by the Council.

Mr. Roberts further proposed that "The Report of the Care and Employment Committee, if accepted and passed by the Council, be published in a journal." This was also agreed to.

The Honorary Secretary then presented the Report on Care and Employment. He pointed out the difficulties in arranging meetings of members of the committee and that the scope of the subject made it necessary to cover a good deal of the ground which had been dealt with in previous reports. It was felt, however, that a comprehensive review of the position must be made in view of the important suggestions incorporated in the report. He then dealt with the main subdivisions of the report and laid particular stress on the sections dealing with the subdivision of the tuberculous population and the cost of tuberculosis to the country as a whole.

He pointed out that a new term—"relative rehabilitation"—had been introduced, and that the proposals embodied in the summary and conclusions were those which had been arrived at as a result of some twenty years of experience in this subject, and that there was strong evidence in the report to substantiate these conclusions.

Dr. Heaf then dealt with that aspect of the report which covered Care Committees, and regretted that it was not possible to include any particular scheme from any one local authority.

In the general discussion which followed there was some criticism of the non-inclusion of the Manchester Scheme, and Dr. Lissant Cox pointed out that the views which had been incorporated in the report and which were attributed to him (*e.g.*, in his 1927 Lancashire Report) were now too old history to be taken as evidence in present-day circumstances.

Dr. Jessel, Dr. Lissant Cox and others felt that too much importance had been laid on the value of voluntary Care Committees, and that as time went on the value of such committees would gradually decrease.

Professor Tytler desired that further stress should be given to the question of the employment of patients in sanatoria and he felt that an index should be added to the report.

After further discussion it was *Resolved* that the report be referred back to the committee and that a special meeting be called to consider the final draft prior to publication.

6. Dr. H. G. Trayer gave an interim report on a questionnaire which had been sent out to over one hundred tuberculosis institutions on the question of shortage of nursing and domestic staff.

He made the significant observation that some 70 per cent. of institutions were short of staff, and that the repercussions would certainly be great if something were not done to alleviate the situation.

The Council *Resolved* that the figures, which were still coming in, be summarised and that the Nursing Committee prepare a final report and circulate it to members of the Council prior to forwarding to the Ministry of Health.

7. Brief consideration was given to a memorandum on "Tuberculosis after the War" prepared by Dr. Ernest Ward and circulated. It was pointed out that a considerable part of this memorandum had been printed in the spring number of the *Bulletin of the National Association for the Prevention of Tuberculosis* and that it could not be considered by the Council in its present form.

8. *Date of Next Meeting.*—The next meeting is to be held in London on the third Saturday in November or on the Saturday following the usual meeting of the Society of Medical Officers of Health.

BOOK REVIEW

Surgery of the Heart. By E. S. J. King, M.D., M.S., D.Sc. (Melb.), F.R.C.S. (Eng.), F.R.A.C.S., Major A.A.M.C., Honorary Surgeon to Out-patients, Royal Melbourne Hospital. London: Edward Arnold and Co. 1941. Price 50s.

This beautifully produced volume of some seven hundred pages covers the surgery of the heart in a comprehensive, if somewhat academic, way. It is replete with illustrations, photographs of specimens, and references, and it deals with rarities as impartially as with common forms of heart disease. The detail is impressive, but it has to be admitted that much of what the author has to say properly belongs to medicine rather than to surgery; and much of what he has to say here is not in line with modern cardiological thought. For instance, his statement that toxic myocarditis due to severe general infections is of great importance to the surgeon, since a post-operative infection or exacerbation of a previous infection may result in a degree of myocardial damage which is quickly followed by cardiac failure, is, to say the least, wanting proof as a common occurrence. Also the remark that angina pectoris has tonsillar infection as a predisposing cause cannot be accepted. This kind of statement mars the book, unfortunately, and it leaves the impression that neither the sifting of the literature nor the medical experience of the writer in heart disease has been critical enough for the task which he set himself in writing this book.

The truth is really that the surgery of the heart is the physician's job, excepting only the purely technical detail of operative procedure. In no field is it so important for the medical aspect of the case to take precedence over everything else, for the opportunity for effective cardiac surgery is rare, so rare that, if we except injuries, constrictive pericarditis, patent ductus, and a few examples of angina and of cardiac failure, it is almost non-existent. Thus the choice of suitable patients, even within these groups, must rest upon a wide experience of cardiology, an experience which is denied to surgeons. A book of this kind adds little to the physician's knowledge; and one is left with the feeling that much more has been made of the subject than is warranted by intrinsic merit.

C. H.

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